Beneficial role of amantadine in catatonic schizophrenia: A case report

Srivastava M

ABSTRACT

Novel pharmacological agents are being used alternatively to manage schizophrenia. However, a significant number of patients do not achieve full remission and have recurrent episodes. One hypothesis illustrates that as a result of pathological processes in the brain, excess glutamate is produced that leads to sequence of events resulting in damage of neurons. NMDA receptor antagonist is proving to be a viable option. It plays a role in reversing the whole sequence. A case report is being presented to highlight its efficacy in treatment of catatonic schizophrenia.

Keywords: amantadine, catatonia, NMDA receptors, receptor antagonist

INTRODUCTION

The use of typical and atypical antipsychotics has offered marked improvement in many schizophrenic patients. However, a significant number of patients do not achieve full remission and have recurrent episodes.[1] Adjunctive therapies have emerged in a big way to tackle the magnitude of schizophrenia. NMDA (N-methyl d-aspartate) receptors have also been targeted for treatment.[2]

Catatonic schizophrenia forms a subtype of schizophrenia, which is often difficult to manage. It has been reported that the use of amantadine in patients with catatonia who do not respond to lorazepam is encouraging.[2] The NMDA receptor antagonism is considered as the mode of action. Memantine, another NMDA antagonist has also been shown to be beneficial in catatonic schizophrenia.[1,3] Documentation on the beneficial results of amantadine is very few.[2] To highlight its beneficial role in catatonia a case report is being presented.

CASE HISTORY

A 24 year old married woman presented to outpatient psychiatry services with mutism, staring, puckering of mouth and ambivalence. She was diagnosed to be case of catatonia and was rated on Bush catatonia scale, her score was 16.[4] She responded well to IV lorazepam 2mg, within half an hour. She started talking adequately to the examiner and smiled. The family refused for admission; hence she was prescribed lorazepam 6mg/day in divided doses, and sent home.

Two days later she reported again with recurrence of symptoms and refused to eat and drink, this time the Bush catatonia score was escalated to 18. She was admitted, all necessary investigations like CT scan, EEG, regular biochemical parameters and thyroid estimation was done and found to be within normal limits. She was switched on to 8 mg/day I.V. lorazepam, in four divided doses. The patient showed no change in symptoms rather, after 2 days became excessively sedated.

Lorazepam was reduced to 6 mg/day, and resperidone in the dose of 4mg/day was added. One week trial of medication showed no change in the symptoms. ECT was prescribed and four ECTs were given on alternate days, however, no change in the catatonia rating scale score was observed. After 10 days, the patient was put on lorazepam 3mg/day in divided doses, and amantadine in the dose of 100 mg, twice daily was added. Next day the patient started taking oral feeds; Ryle's tube could be removed on the 4th day, the Bush catatonia rating score decreased to 6 in a week's time. One week later the amantadine was increased to 150mg, twice daily.

The patient was discharged. Follow ups were regular and the patient integrated well into the family. Six weeks later lorazepam was tapered and withdrawn. She became a productive member of her family within a year and is maintaining well on 200mg/day of amantadine.

DISCUSSION

Amantadine has NMDA receptor antagonist

activity, in addition to its dopamine agonist activity.[2] It is approved for use in Parkinson's disease and extra pyramidal diseases.[2] Its potential efficacy in catatonia may be due to blockade of hyperglutaminergic excitotoxicity activity in neurons.[2,3] It is hypothesized that as a result of the pathological process in the brain, excess glutamate is produced.[2] Increase in glutamate leads to hyperexcitation of the respective receptors which indirectly allow the calcium channel to remain open. Calcium influx leads to free radical formation and therefore causes neuronal damage.[2,3] This whole process is reversed by amantadine for a longer duration.[1] More researches are required to understand the efficacy of NMDA receptor antagonists as safe treatment options for patients of catatonia.

AUTHOR NOTE

Mona Srivastava, Contact- 9336910619,
Email: drmonasrivastava@gmail.com
(Corresponding Author)
Department of psychiatry, Institute of Medical Sciences, Banaras Hindu University, Varanasi-5

REFERENCES

1. Carroll BT, Thomas C, Jayanti K. Amantadine and memantine in catatonic schizophrenia. *Ann Clin Psychiatry.* 2006 Apr-Jun;18(2):133-4.
2. Goff DC, Coyle JT. The emerging role of glutamate in the pathophysiology and treatment of schizophrenia. *Am J Psychiatry.* 2001;158(9):1376-77.
3. Northoff G. What catatonia can tell us about “top-down modulation”: a neuropsychiatric hypothesis. *Behave Brain Sci.* 2002;25(5):555-77.
4. Bush G, Fink M, Petrides G, Dowling F, Francis A. Catatonia. I. Rating scale and standardized examination. *Acta Psychiatr Scand.* 1996 Feb;93(2):129-36.